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INDEX 'AIDSLINE, ANABSTR, [illegible]ASCI, BIOBUSINESS, BIOSIS, [illegible]IOTECHABS, BIOTECHDS, CABA, CANCERLIT, CAPLUS, CEABA, CEN, CIN, CJACS, CJELSEVIER, CONFSCI, DDFB, DDFU, DISSABS, DRUGB, DRUGLAUNCH, DRUGNL, DRUGU, EMBAL, EMBASE, FSTA, GENBANK, HEALSAFE, IFIPAT, ...' ENTERED AT 09:40:36 ON 25 JAN 96

42 FILES IN THE FILE LIST IN STNINDEX

L1 QUE GLUTAMATE AND TRANSPORTER# AND (CLONE# OR DNA# OR CDNA#)
L15 475 L1
L30 192 L15 AND HUMAN
L31 79 DUPLICATE REMOVE L30
L31 ANSWER 50 OF 79 CANCERLIT DUPLICATE 22
94086549 Phosphorylation and modulation of brain ***glutamate*** ***transporters*** by protein kinase C. Casado M; Bendahan A; Zafra F; Danbolt N C; Aragon C; Gimenez C; Kanner B I. Centro de Biologia Molecular, Facultad de Ciencias, Universidad Autonoma de Madrid, Spain.. J Biol Chem (1993). Vol. 268, No. 36, pp. 27313-7. Journal code: HIV. ISSN: 0021-9258. Language: English.
AB High affinity sodium- and potassium-coupled L- ***glutamate*** transport into presynaptic nerve terminals and fine glial processes removes the neurotransmitter from the synaptic cleft, thereby terminating glutamergic transmission. This report describes that the purified L- ***glutamate*** ***transporter*** from pig brain is phosphorylated by protein kinase C, predominantly at serine residues. Upon exposure of C6 cells, a cell line of glial origin, to 12-O-tetradecanoylphorbol-13-acetate, about a 2-fold stimulation of L- ***glutamate*** transport is observed within 30 min. Concomitantly, the level of phosphorylation increases with similar kinetics. The phorbol ester also stimulates L- ***glutamate*** transport in HeLa cells infected with a recombinant vaccinia virus expressing T7 RNA polymerase and transfected with pT7-GLT-1. The latter is a recently ***cloned*** rat brain ***glutamate*** ***transporter*** of glial origin. Mutation of serine 113 to asparagine does not affect the levels of expressed transport but abolishes its stimulation by the phorbol ester. To our knowledge, this is the first direct demonstration of the regulation of a neurotransmitter ***transporter*** by phosphorylation.

L31 ANSWER 51 OF 79 BIOSIS COPYRIGHT 1996 BIOSIS DUPLICATE 23
93:412930 Document No.: BA96:78655. CLONING AND EXPRESSION OF A NOVEL SODIUM DEPENDENT NEUTRAL AMINO ACID ***TRANSPORTER*** STRUCTURALLY RELATED TO MAMMALIAN SODIUM- ***GLUTAMATE*** COTRANSPORTERS. SHAFQAT S; TAMARAPPOO B K; KILBERG M S; PURANAM R S; MCNAMARA J O; GUADANO-FERRAZ A; FREMEAU R T JR. DEP. PHARMACOL., BOX 3813, DUKE UNIVERSITY MED. CENTER, 436 NANALINE H. DUKE BUILDING, RES. DR., DURHAM, NC 27710, USA. J BIOL CHEM, 268 (21). 1993. 15351-15355. CODEN: JBCHA3; ISSN: 0021-9258. Language: English
AN 93:412930 BIOSIS
AB A ***cDNA*** has been isolated from ***human*** hippocampus that appears to encode a novel Na+-dependent, Cl-independent, neutral amino acid ***transporter*** . The putative protein, designated SATT, is 529 amino acids long and exhibits significant amino acid sequence identity (3944%) with mammalian L- ***glutamate*** ***transporters*** . Expression of SATT ***cDNA*** in HeLa cells induced stereospecific uptake of L-serine, L-alanine, and L-threonine that was not inhibited by excess (3 mM) 2-(methylamino)-isobutyrie acid, a specific substrate for the System A amino acid ***transporter*** . SATT expression in HeLa cells did not induce the transport of radiolabeled L-cysteine, L- ***glutamate*** , or related dicarboxylates. Northern blot hybridization revealed high levels of SATT mRNA in ***human*** skeletal muscle, pancreas, and brain, intermediate levels in heart, and low levels in liver,

placenta, lung, and kidney. SATT transport characteristics are similar to the Na+-dependent neutral amino acid transport activity designated System ASC, but important differences are noted. These include: 1) SATT's apparent low expression in ASC-containing tissues such as liver or placenta; 2) the lack of mutual inhibition between serine and cysteine; and 3) the lack of trans-stimulation. SATT may represent one of multiple activities that exhibit System ASC-like transport characteristics in diverse tissues and cell lines.

L31 ANSWER 52 OF 79 BIOSIS COPYRIGHT 1996 BIOSIS DUPLICATE 24
93:412931 Document No.: BA96:78656. CLONING AND EXPRESSION OF A ***HUMAN*** NEUTRAL AMINO ACID ***TRANSPORTER*** WITH STRUCTURAL SIMILARITY TO THE ***GLUTAMATE*** ***TRANSPORTER*** GENE FAMILY. ARRIZA J L; KAVANAUGH M P; FAIRMAN W A; WU Y-N; MURDOCH G H; NORTH R A; AMARA S G. OREGON HEALTH SCI. UNIVERSITY, VOLLUM INST. L-474, 3181 SW SAM JACKSON PARK RD., PORTLAND, OR 97201-3098, USA. J BIOL CHEM, 268 (21). 1993. 15329-15332. CODEN: JBCHA3; ISSN: 0021-9258. Language: English
AN 93:412931 BIOSIS
AB A ***cDNA*** was isolated from ***human*** brain that encodes an amino acid sequence 34-39% identical to previously published ***glutamate*** ***transporter*** sequences. Injection of RNA transcribed from this ***cDNA*** into Xenopus oocytes resulted in expression of a transport activity with the properties of the neutral amino acid uptake system ASC. Superfusion of alanine, serine, and cysteine evoked sodium-dependent inward currents in voltage-clamped oocytes expressing the ***transporter*** . These currents were dose-dependent, stereospecific, and saturable, with Km values ranging from 29 to 88 .mu.M. Northern blot analyses revealed ubiquitous expression of this gene, termed ASCT1, consistent with the general metabolic role ascribed to system ASC.

L31 ANSWER 57 OF 79 BIOSIS COPYRIGHT 1996 BIOSIS
94:80293 Document No.: 97093293. A new family of neurotransmitter ***transporter*** : The high-affinity ***glutamate*** ***transporters*** .. Kanai Y; Smith C P; Hediger M A. Renal Div., Dep. Med., Brigham Women's Hosp., Harvard Med. Sch., Boston, MA 02115, USA FASEB (Federation of American Societies for Experimental Biology) Journal. 7 (15). 1993. 1450-1459. ISSN: 0892-6638. Language: English
AN 94:80293 BIOSIS
AB An essential component of the transmission process at glutamatergic synapses is the removal of ***glutamate*** from the synaptic deft. This is achieved by powerful transport systems which have a high affinity for ***glutamate*** and exhibit a novel coupling to inorganic ions. ***Transporters*** situated on presynaptic termini sequester ***glutamate*** directly from the synaptic cleft. In concert, ***transporters*** situated on glial cells maintain a low extracellular ***glutamate*** concentration, thereby establishing a diffusion gradient favoring movement of ***glutamate*** out of the synaptic cleft. Maintenance of a low extracellular ***glutamate*** concentration also serves to protect neurons from the excitotoxic action of ***glutamate*** . Despite the physiological importance of the ***glutamate*** ***transporters*** , little information has been available on their molecular structures. This gap, however, has begun to be bridged with the recent cloning of three species of eukaryotic ***glutamate*** ***transporters*** . The purpose of this review is to summarize the results of these three cloning successes, to compare and contrast the three novel ***transporters*** , and to reinterpret, in the light of these recent breakthroughs, information from previous studies.

L31 ANSWER 60 OF 79 BIOSIS COPYRIGHT 1996 BIOSIS

93:267855 Document No.: [redacted]4:130005. CLONING AND CHARACTERIZATION OF ***HUMAN*** HIGH AFFINITY ***GLUTAMATE*** [redacted]*TRANSPORTER*** ***CDNAS*** . STELZNER M; SMITH C P; KANAI Y; KHAWAJA S; BOUTIN P M; HEDIGER M A. RENAL DIV., DEP. MED., BRIGHAM AND WOMEN'S HOSP., HARVARD MED. SCH., BOSTON, MA 02115, USA. MEETING OF THE FEDERATION OF AMERICAN SOCIETIES FOR EXPERIMENTAL BIOLOGY ON EXPERIMENTAL BIOLOGY '93, NEW ORLEANS, LOUISIANA, USA, MARCH 28-APRIL 1, 1993. FASEB (FED AM SOC EXP BIOL) J, 7 (3-4). 1993. A575. CODEN: FAJOEC; ISSN: 0892-6638. Language: English
AN 93:267855 BIOSIS

L31 ANSWER 61 OF 79 SCISEARCH COPYRIGHT 1996 ISI (R)
93:146293 The Genuine Article (R) Number: KP975. CLONING AND CHARACTERIZATION OF ***HUMAN*** HIGH-AFFINITY ***GLUTAMATE*** ***TRANSPORTER*** ***CDNAS*** . STELZNER M (Reprint); SMITH C P; KANAI Y; KHAWAJA S; BOUTIN P M; HEDIGER M A. HARVARD UNIV, BRIGHAM & WOMENS HOSP, SCH MED, DEPT MED, DIV RENAL, BOSTON, MA, 02115. FASEB JOURNAL (23 FEB 1993) Vol. 7, No. 4, Part 2, pp. A575. ISSN: 0892-6638. Pub. country: USA. Language: ENGLISH.
QH 301, F4 ✓

L31 ANSWER 62 OF 79 BIOSIS COPYRIGHT 1996 BIOSIS
94:5574 Document No.: 97018574. Pharmacological characterization of ***cloned*** ***human*** ***glutamate*** ***transporter*** subtypes.. Fairman W A; Arriza J L; Amara S G. Vollum Inst., Oregon Health Sci. Univ., Portland, OR 97201, USA Society for Neuroscience Abstracts23rd Annual Meeting of the Society for Neuroscience, Washington, D.C., USA, November 7-12, 1993., 19 (1-3). 1993. 496. ISSN: 0190-5295. Language: English
AN 94:5574 BIOSIS

L31 ANSWER 63 OF 79 EMBASE COPYRIGHT 1996 ELSEVIER SCI. B.V.DUPLICATE 26
93165884 EMBASE Excitatory amino acid receptors, excitotoxicity and the ***human*** nervous system. Shaw P.J.. Department of Neurology, Ward G, Royal Victoria Infirmary, Newcastle upon Tyne NE1 4LP, United Kingdom. CURR. OPIN. NEUROL. NEUROSURG. 6/3 (414-422) 1993. ISSN: 0951-7383. CODEN: CNENE8. Pub. Country: United Kingdom. Language: English. Summary Language: English.
✓ date? ordered 1/25
AB ***Glutamate*** receptors continued to be the subject of intense investigation during 1992. It is clear that there is great structural and functional diversity within this receptor family, although the precise subunit structure of excitatory amino acid (EAA) receptors in specific neuronal groups within the ***human*** central nervous system remains to be determined. Molecular studies have shown the existence of five genes encoding N-methyl-D-aspartate receptor subunits that have specific anatomic profiles and differing functional properties. The chromosomal localization of several genes encoding EAA receptor subunits has been established and some of these represent candidate genes for clinical neurologic disorders. Further insights were gained into the functions of metabotropic receptors, and three distinct genes encoding ***glutamate*** ***transporters*** were ***cloned*** . The interaction between neurotrophic factors and EAA neurotransmitters is increasingly recognized. Excitotoxicity is considered to represent a final common pathway of neuronal injury in an ever-increasing range of neurologic disorders. The development of therapeutic agents has focused on methods for reducing excitotoxicity without interfering with EAA receptor activation.

L31 ANSWER 64 OF 79 MEDLINE
94054671 The elusive ***transporters*** with a high affinity for ***glutamate*** . Kanai Y; Smith C P; Hediger M A. (Dept of Medicine, Brigham and Women's Hospital, Boston, MA.). Trends

Neurosci, (1993 Sep) 1[illegible](9) 365-70. Ref: 48. Journal code: WEL. ISSN: 0378-5912. Pub. Country: ENGLAND: United Kingdom. Language: English.

QP351.T69

AB Removal of ***glutamate*** from the synaptic cleft is an essential component of the transmission process at glutamatergic synapses. This requirement is fulfilled by ***transporters*** that have a high affinity for ***glutamate*** and exhibit a unique coupling to Na+, K+ and OH- ions. Independently, three groups have succeeded in cloning ***cDNAs*** encoding high-affinity Na(+)-dependent ***glutamate*** ***transporters*** . These ***transporters*** are structurally distinct from previously characterized neurotransmitter ***transporters*** and show sequence identity with prokaryotic ***glutamate*** and dicarboxylate ***transporters*** . In addition, they exhibit significant differences in their structure, function and tissue distribution. This review compares and contrasts these differences, and incorporates into the existing body of knowledge these new breakthroughs.

L31 ANSWER 66 OF 79 BIOSIS COPYRIGHT 1996 BIOSIS DUPLICATE 27
94:17745 Document No.: 97030745. Cloning and characterization of a ***glutamate*** ***transporter*** ***cDNA*** from ***human*** cerebellum.. Shashidharan P; Plaitakis A. Dep. Neurol., Mt. Sinai Sch. Med., One Gustave L. Levy Pl., New York, NY 10029, USA Biochimica et Biophysica Acta, 1216 (1). 1993. 161-164. ISSN: 0006-3002. Language: English

date? QD1.B5

AN 94:17745 BIOSIS
AB The ***glutamate*** /aspartate ***transporters*** are essential for the elimination and recycling of synaptic ***glutamate*** released from nerve endings during neurotransmission. Evidence suggests that these processes are altered in ischemia and neuronal degenerations linked to excitotoxicity. We screened a ***cDNA*** library constructed from ***human*** cerebellar mRNA, and isolated a ***cDNA*** that shows an 88.5% and a 98.7% sequence similarity at the nucleotide and amino acid level, respectively, with a rat brain specific Na+-dependent ***glutamate*** /aspartate ***transporter*** . The ***human*** ***cDNA*** is expressed in brain and it may prove useful in the study of ***human*** neurodegenerations linked to ***glutamate*** dysfunction.

L44 36 (EXITATORY(W) AMINO(W) ACID(W) TRANSPORTER(W)(2 OR II)) OR EAAT2 OR (GLUTAMATE(W) TRANSPORTER(W)(2 OR II))
L45 9 DUPLICATE REMOVE L44
L45 ANSWER 1 OF 9 BIOSIS COPYRIGHT 1996 BIOSIS DUPLICATE 1
95:403582 Document No.: 98417882. Constitutive Ion Fluxes and Substrate Binding Domains of Human Glutamate Transporters.. Vandenberg R J; Arriza J L; Amara S G; Kavanaugh M P. Vollum Inst., Oregon Health Sci. Univ., Portland, OR 97201, USA Journal of Biological Chemistry, 270 (30). 1995. 17668-17671. ISSN: 0021-9258. Language: English
AN 95:403582 BIOSIS
AB Application of L-glutamate activates ionic currents in voltage-clamped Xenopus oocytes expressing cloned human excitatory amino acid transporters (EAATs). However, even in the absence of L-glutamate, the membrane conductance of oocytes expressing EAAT1 was significantly increased relative to oocytes expressing ***EAAT2*** or control oocytes. Whereas transport mediated by ***EAAT2*** is blocked by the non-transported competitive glutamate analog kainate (K-i = 14 mu-M), EAAT1 is relatively insensitive (K-i gt 3 mM). Substitution of a block of 76 residues from ***EAAT2*** into EAAT1, in which 18 residues varied from EAAT1, conferred high affinity kainate binding to EAAT1, and application of kainate to

oocytes expressing the chimeric transporter blocked a pre-existing monovalent cation conductance that displayed a permeability sequence K+ gt Na+ gt Li+ mchgt choline+. The results identify a structural domain of glutamate transporters that influences kainate binding and demonstrate the presence of a constitutive ion-selective pore in the transporter.

L45 ANSWER 2 OF 9 BIOSIS COPYRIGHT 1996 BIOSIS DUPLICATE 2
95:209841 Document No.: 98224141. Differential modulation of human glutamate transporter subtypes by arachidonic acid.. Zerangue N; Arriza J L; Amara S G; Kavanugh M P. Vollum Inst., Oregon Health Sci. Univ., 3181 Sam Jackson Pk. Road, Portland, OR 97201, USA Journal of Biological Chemistry, 270 (12). 1995. 6433-6435. ISSN: 0021-9258. Language: English
AN 95:209841 BIOSIS
AB Arachidonic acid has been proposed to be a messenger molecule released following synaptic activation of glutamate receptors and during ischemia. Here we demonstrate that micromolar levels of arachidonic acid inhibit glutamate uptake mediated by EAAT1, a human excitatory amino acid transporter widely expressed in brain and cerebellum, by reducing the maximal transport rate approximately 30%. In contrast, arachidonic acid increased transport mediated by ***EAAT2*** , a subtype abundantly expressed in forebrain and midbrain, by causing the apparent affinity for glutamate to increase more than 2-fold. The results demonstrate that the response of different glutamate transporter subtypes to arachidonic acid could influence synaptic transmission and modulate excitotoxicity via positive or negative feedback according to the transporters present in a particular region.

L45 ANSWER 3 OF 9 EMBASE COPYRIGHT 1996 ELSEVIER SCI. B.V.
95318511 EMBASE Glutamate-gated chloride channel with glutamate-transporter-like properties in cone photoreceptors of the tiger salamander. Picaud S.A.; Larsson H.P.; Grant G.B.; Lecar H.; Werblin F.S.. Life Science Addition 145, University of California, Berkeley, CA 94720, United States. Journal of Neurophysiology 74/4 (1760-1771) 1995. ISSN: 0022-3077. CODEN: JONEA4. Pub. Country: United States. Language: English. Summary Language: English.
AB 1. Using the patch-clamp technique, we investigated whether the glutamate elicited current in mechanically isolated cone photoreceptors from the salamander retina is generated by a Cl- channel or a ***glutamate*** ***transporter*** . ***2*** . The current reversed near the equilibrium potential for Cl-, was decreased by three Cl- channel blockers, 5-nitro-2-(3-phenyl-propylamino) benzoic acid, 4,4'-diisothiocyanostilbene-2,2'-disulfonate, and diphenylamine 2,2'-dicarboxylic acid, and was eliminated when gluconate was substituted for both internal and external Cl-, features consistent with the current being mediated by a Cl- channel. 3. The single-channel conductance of the Cl- channel was estimated by noise analysis of the glutamate-elicited current fluctuations to be 0.7 pS with an open time of 2 ms. 4. The magnitude of the current was dependent on both internal and external Na+ and K+, features consistent with the current being related to the activation of a glutamate transporter. Yet changes in their concentrations did not affect the reversal potential of the current. 5. Taken together with earlier reports on this current showing that it has a glutamate-transporter-like pharmacology, our results suggest that the glutamate elicited current is carried by a Cl- channel but gated by a glutamate receptor whose pharmacology and ionic requirement resemble those previously described for glutamate transporters.

L45 ANSWER 4 OF 9 CAPLUS COPYRIGHT 1996 ACS

1995:841609 Document No. [redacted]3:247405 Ion fluxes asso[redacted]ated with excitatory amino acid transport. Wadiche, Jacque[redacted].; Amara, Susan G.; Kavanaugh, Michael P. (Vollum Inst., Oregon Health Sci. Univ., Portland, OR, 97201, USA). Neuron, 15(3), 721-8 (English) 1995. CODEN: NERNET. ISSN: 0896-6273.

AB Flux of substrate and charge mediated by three cloned excitatory amino acid transporters widely expressed in human brain were studied in voltage-clamped Xenopus oocytes. Superfusion of L-glutamate or D-aspartate resulted in currents due in part to electrogenic Na+ cotransport, which contributed 1 net pos. charge per transport cycle. A significant addnl. component of the currents was due to activation of a reversible anion flux that was not thermodynamically coupled to amino acid transport. The selectivity sequence of this ligand-activated conductance was NO3- > I- > Br- > Cl- > F-. The results suggest that these proteins mediate both transporter- and channel-like modes of permeation, providing a potential mechanism for dampening cell excitability, in addn. to removal of transmitter.

L45 ANSWER 5 OF 9 BIOSIS COPYRIGHT 1996 BIOSIS DUPLICATE 3

95:510748 Document No.: 98515798. Molecular characterization of a high-affinity mouse glutamate transporter.. Sutherland M L; Delaney T A; Noebels J L. Dep. Neurology, Baylor Coll. Med., One Baylor Plaza, Houston, TX 77030, USA Gene (Amsterdam), 162 (2). 1995. 271-274. ISSN: 0378-1119. Language: English

AN 95:510748 BIOSIS

AB The complete coding sequence of a mouse glutamate transporter (mEAAT2) has been cloned by polymerase chain reaction (PCR) from adult whole-brain total RNA. Southern hybridization analysis of PCR products amplified from templates derived from various murine adult tissues demonstrated that the transcript for mEAAT2 was specific to the central nervous system. High-affinity transport of D-aspartate, K-m value (17 +- 5 mu-M), was determined in a vaccinia/T7 RNA polymerase expression system. The deduced amino-acid sequence of mEAAT2 shares 96 and 93% identity with the rat and human ***EAAT2*** homologues, respectively.

L45 ANSWER 6 OF 9 BIOSIS COPYRIGHT 1996 BIOSIS DUPLICATE 4

95:548567 Document No.: 98562867. Assignment of the gene SLC1A2 coding for the human glutamate transporter ***EAAT2*** to human chromosome 11 bands p13-p12.. Li X; Francke U. Howard Hughes Med. Inst., Stanford Univ. Med. Cent., Stanford, CA 94305-5428, USA Cytogenetics and Cell Genetics, 71 (3). 1995. 212-213. ISSN: 0301-0171. Language: English

AN 95:548567 BIOSIS

L45 ANSWER 7 OF 9 BIOSIS COPYRIGHT 1996 BIOSIS DUPLICATE 5

94:503020 Document No.: 97516020. Functional comparisons of three glutamate transporter subtypes cloned from human motor cortex.. Arriza J L; Fairman W A; Wadiche J I; Murdoch G H; Kavanaugh M P; Amara S G. Oregon Health Sci. Univ., Vollum Institute L-474, 3181 SW Sam Jackson Park Road, Portland, OR 97201-3098, USA Journal of Neuroscience, 14 (9). 1994. 5559-5569. ISSN: 0270-6474. Language: English

AN 94:503020 BIOSIS

AB Reuptake plays an important role in regulating synaptic and extracellular concentrations of glutamate. Three glutamate transporters expressed in human motor cortex, termed EAAT1, ***EAAT2*** , and EAAT3 (for excitatory amino acid transporter), have been characterized by their molecular cloning and functional expression. Each EAAT subtype mRNA was found in all human brain regions analyzed. The most prominent regional variation in message content was in cerebellum where EAAT1 expression predominated. EAAT1 and EAAT3 mRNAs were also expressed in various non-nervous tissues,

whereas expression of ***EAAT2*** was largely restricted to brain. The kinetic parameters and pharmacological characteristics of transport mediated by each EAAT subtype were determined in transfected mammalian cells by radiolabel uptake and in microinjected oocytes by voltage-clamp measurements. The affinities of the EAAT subtypes for L-glutamate were similar, with K-m determinations varying from 48 to 97 mu-M in the mammalian cell assay and from 18 to 28 Am in oocytes. Glutamate uptake inhibitors were used to compare the pharmacologies of the EAAT subtypes. The ***EAAT2*** subtype was distinguishable from the EAAT1/EAAT3 subtypes by the potency of several inhibitors, but most notably by sensitivity to kainic acid (KA) and dihydrokainic acid (DHK). KA and DHK potently inhibited ***EAAT2*** transport, but did not significantly affect transport by EAAT1/EAAT3. Using voltage-clamp measurements, most inhibitors were found to be substrates that elicited transport currents. In contrast, KA and DHK did not evoke currents and they were found to block ***EAAT2*** -mediated transport competitively. This selective interaction with the ***EAAT2*** subtype could be a significant factor in KA neurotoxicity. These studies provide a foundation for understanding the role of glutamate transporters in human excitatory neurotransmission and in neuropathology.

L45 ANSWER 8 OF 9 CAPLUS COPYRIGHT 1996 ACS
1995:58824 Document No. 122:124663 The mouse and human excitatory amino acid transporter gene (EAAT1) maps to mouse chromosome 15 and a region of syntenic homology on human chromosome 5. Kirschner, M. A.; Arriza, J. L.; Copeland, N. G.; Gilbert, D. J.; Jenkins, N. A.; Magenis, E.; Amara, S. G. (Vollum Inst. Adv. Biomed. Res., Oregon Health Sciences Univ., Portland, OR, 97201, USA). Genomics, 22(3), 631-3 (English) 1994. CODEN: GNMCEP. ISSN: 0888-7543.
AB The gene for human excitatory amino acid transporter (EAAT1) was localized to the distal region of human chromosome 5p13 by in situ hybridization of metaphase chromosome spreads. Interspecific backcross anal. identified the mouse Eaat1 locus in a region of 5p13 homol. on mouse chromosome 15. Markers that are linked with EAAT1 on both human and mouse chromosomes include the receptors for leukemia inhibitory factor, interleukin-7, and prolactin. The Eaat1 locus appears not to be linked to the epilepsy mutant stg locus, which is also on chromosome 15. The EAAT1 locus is located in a region of 5p deletions that have been assocd. with mental retardation and microcephaly.

L45 ANSWER 9 OF 9 BIOSIS COPYRIGHT 1996 BIOSIS DUPLICATE 6
95:77557 Document No.: 98091857. Mouse excitatory amino acid transporter ***EAAT2*** : Isolation, characterization, and proximity to neuroexcitability loci on mouse chromosome 2.. Kirschner M A; Copeland N G; Gilbert D J; Jenkins N A; Amara S G. Vollum Inst. Advanced Biomedical Res., Dep. Neurol., Oregon Health Sciences University, 3181 Sam Jackson Park Road, Portland, OR 97201, USA Genomics, 24 (2). 1994. 218-224. ISSN: 0888-7543. Language: English
AN 95:77557 BIOSIS
AB Glutamate and aspartate are excitatory neurotransmitters that have been implicated in a number of pathological states of the nervous system. Accumulation of extracellular excitatory amino acids can be cytotoxic and may also lower the seizure threshold in epilepsy. An important function of the Na+-dependent high-affinity excitatory amino acid transporter (EAAT) is the reuptake of secreted amino acid neurotransmitter, possibly maintaining extracellular amino acid concentrations at nontoxic and nonepileptogenic levels. We have isolated the mouse cDNA for ***EAAT2*** , a neurotransmitter transporter that shares extensive amino acid sequence homology with one of several previously cloned high-affinity glutamate

transporters. The mouse ***EAAT2*** amino acid sequence shares 99 and 97% identity with the rat and human homologues, respectively. It is expressed predominantly in the brain, where it may function as a glia-specific transporter. In an interspecific backcross analysis ***Eaat2*** maps to the central region of mouse chromosome 2, where it is located near quantitative trait loci that modulate neuroexcitability and seizure frequency in mouse models of alcohol withdrawal and epilepsy.